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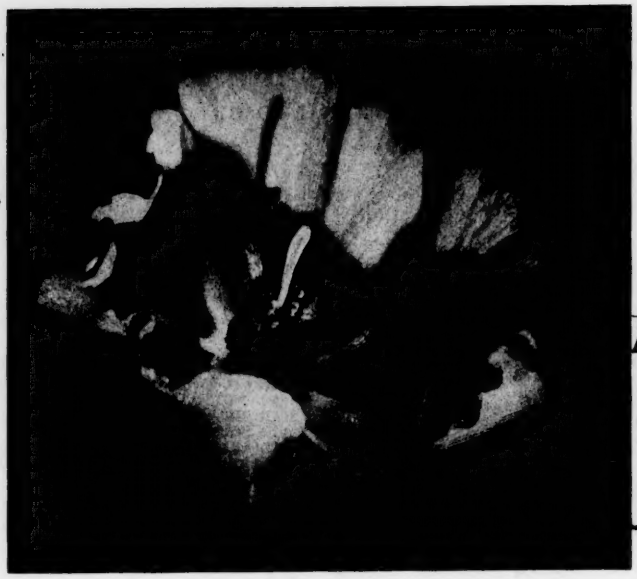

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TRISPECIES COTTON
(SEE PAGE 291)

Mice With Red Eyes and Black

Twins With Identical Spondylitis

Recessive Ichthyosis in the Mouse

Genetics and the Evolution of Plants

Taste-Blindness and Diabetes in Negroes

Punched Cards to Compute Inbreeding

VOLUME 41 NOVEMBER, 1950 NUMBER 11

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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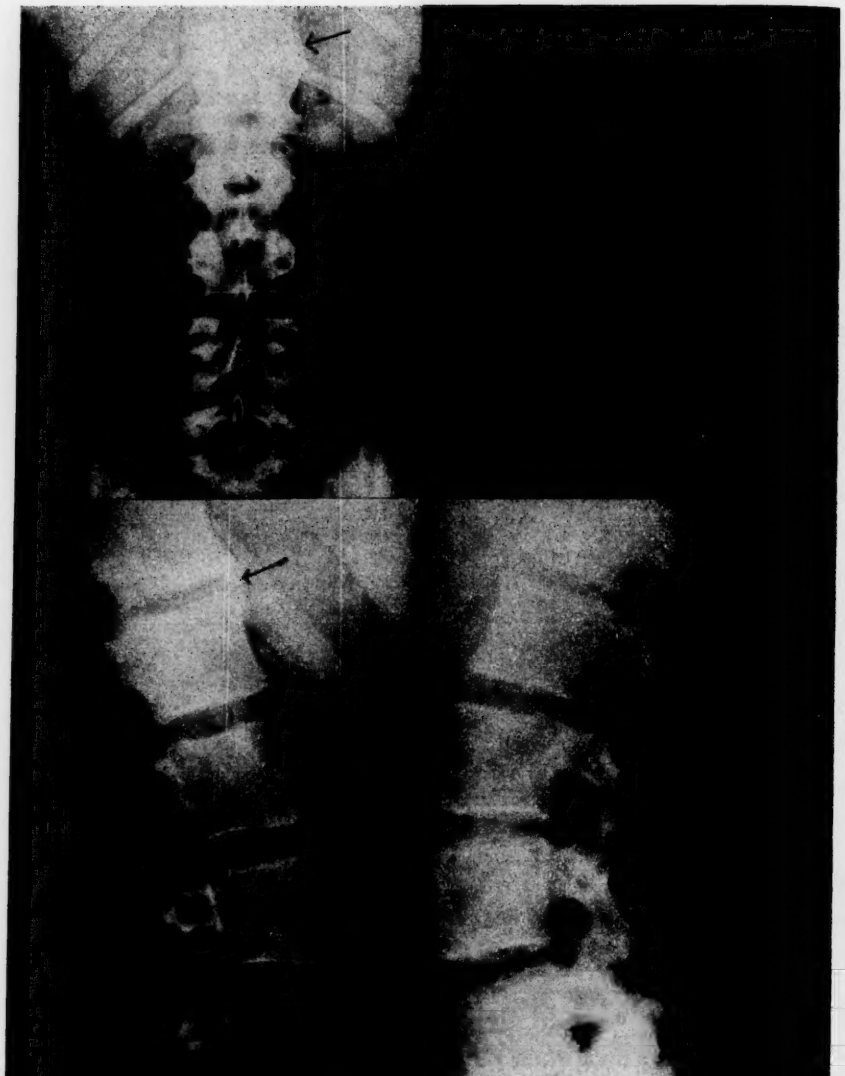

SPINAL AND SACRAL CHANGES IN SEPARATED IDENTICAL TWINS

Figure 1

The two left hand plates show dorsal and lateral views of the spine of twin A, and the two right hand plates show similar photographs of twin B. The arrow in plate A-1 shows syndesmophytes between vertebrae T-12 and L-1 and destructive changes in both sacro-iliac joints. In Twin B (Figure *B*-1) the beginning of syndesmophyte formation is indicated between vertebrae T-10-11-12. Also destructive changes are visible in both sacro-iliac joints. The lateral views (lower plates) show the same formation between vertebrae T-12 and L-1 in both twins as is shown in the upper plate. In both twins the lumbar curve is well preserved with no intervertebral calcification of the majority of the lumbar vertebrae.

SPONDYLITIS IN IDENTICAL TWINS REARED APART*

F. E. Stephens and John C. Nunemaker, M.D.†

In the study of human inheritance, it is seldom possible to supplement pedigree studies with the study of the same trait in identical twins. The present investigation is of special interest, therefore, since it is concerned with observations on spondylitis occurring in both members of a pair of identical twins who have been reared apart since infancy. They had previously been reported by Stephens and Thompson[8] as a typical example of identical twins reared apart. In these two young men the disease not only developed at approximately the same time but the unusual pattern of progress of the disease was almost identical in each.

Since Claussen and Kober[2] and Neel[4] have suggested that other forms of rheumatoid arthritis indicate the presence of predisposing factors for spondylitis, members of the father's and of the mother's families have been carefully investigated for rheumatoid arthritis as well as spondylitis.

Description of the Disease

Rheumatoid spondylitis is a chronic, progressive, crippling disease of the spine which occurs predominantly in males of the age group 20 to 40, and is associated with rheumatoid arthritis of the extremities in 15 to 20 percent of the cases. Although there are reasons for thinking of the disease as different from the peripheral type of rheumatoid arthritis, most rheumatologists prefer to consider them related. Therefore, the term "rheumatoid spondylitis" is preferred to the many other eponymic and descriptive names such as Marie-Strumpell's disease, von Bechterew's disease, spondylitis rhizomelique, spondylarthritis ankylopoietica, poker-spine, and bamboo-spine.

Characteristically, the disease begins in the sacroiliac joints and slowly ascends the spine over a variable period of months or years. If it is not arrested, the disease may progress so far as to preclude motion of the entire spine with eventual calcification of the intervertebral ligaments. Oftentimes, in the early stages, low backache or sciatica-like pain down the legs is the only symptom. In some cases there may be associated loss of motion in the hips or shoulder joints without any other peripheral joint disease. Other than orthopedic exercises, braces and physiotherapeutic manipulation, therapy is usually limited to the use of salicylates and X-ray treatment of the spine.

Review of Literature Pertaining to the Inheritance

Although much has been written on the subject of spondylitis, there are relatively few references to the possible inheritance of a susceptibility to the disease and some of the evidence is not convincing.

Dawson and Ragan[3] reported on 221 cases. Among these were a sister and brother and a sister and two brothers with spondylitis. They called attention to a report by Berckhardt of a family in which the mother and five children, all males, contracted the disease. Stecher and Hauser[7], reviewing the literature dealing with the hereditary aspects of the disease, concluded that only four fully authenticated cases of two brothers with the disease had been reported up to 1946. Among these cases were those reported by Weil and Allolio, Reynolds, and Ehrlich. Stecher and Hauser add another case of two brothers with the disease to this list.

Campbell[1] saw twenty-five cases of the disease. Of these, two were identical twins, two were brothers, one had a brother with spondylitis, and one had an uncle and a brother with the disease. Rogoff and Freyberg[6] quote Ray as having reported the condition in identical twins and Scott as having reported the condition in a brother and sister, two sisters, and a father and son. Rogoff and Freyberg found a father and three sons; a man, his father and his uncle; and a pair of identical twins with

*This study was supported by a grant from the United States Public Health Service. It was reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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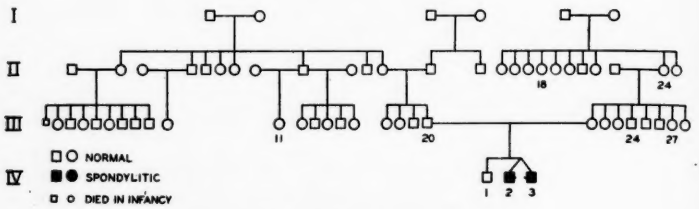

ANCESTRY OF THE AFFECTED TWINS

Figure 2

Some of the twins' ancestors and collateral relatives exhibit rheumatic symptoms, as is explained in the text below. But none suffered from spondylitis.

spondylitis. In a series of 114 cases, they found the familial incidence to be 9 percent or over 100 times greater than in the general population.

Neel[4] reported a kindred in which a woman with spondylitis had thirteen children, three of whom developed the condition. These, in turn, had five children, two of whom were likewise affected. Classen and Kober[2] give ten pedigrees in which spondylitis is accompanied by rheumatic diseases: polyartharitis rheumatica, arthritis, and muscular rheumatism.

Neel[4] suggested an autosomal dominant as the predisposing factor in the kindred he studied. He also suggested that the high incidence of arthritis, other than spondylitis, in this particular kindred was an indication of the presence of modifiers which increase the penetrance of the dominant for spondylitis.

Most authors are agreed that the incidence of spondylitis is particularly great in males. Dawson and Ragan[3] found that about 80 percent of the 221 cases they studied were in males. They point out that the sex ratio for peripheral rheumatoid arthritis is about one affected male to three affected females as opposed to the high incidence of spondylitis in the male. This might be taken as an indication that spondylitis is a disease distinct from other forms of arthritis. In a study of 1,135 cases of spondylitis, Polley and Slocumb[5] derived a ratio of nine affected males to one affected female. Dawson and Ragan[3] quote Ehrlich as reporting that less than 10 percent of 753 cases were in females.

Spondylitis in the Twins and Their Families

Figure 2 shows a pedigree chart of the identical twins (IV-2,3). The brother (IV-1) and the father (III-20) were both examined and no indication of spondylitis, rheumatism, or athritis was found. Interviews with other members of the families indicated no unusual incidence of arthritic disease. One woman (III-11), a cousin of the twin's father, was reported to have had arthritis of the spine. She moved to Arizona where she now lives. She states that since 1945 her back has not given her sufficient trouble to warrant seeing a doctor. In the light of the evidence available, it is very doubtful if she actually has spondylitis.

Several cases of high blood pressure were found on the father's side of the family. The mother of the twins is dead but her mother and sister do not recall her having rheumatic or arthritic symptoms. One of her sisters (III-27) and one brother who was polydactylous (III-24) had had trouble with their backs but physical examinations as well as a roentgenogram of the brother showed no indication of spondylitis. The grandmother (II-24) reported that one of her sisters (II-18) had had arthritis and swollen joints. Other than this, there is little evidence of rheumatism or arthritis of any kind in the family. The grandfather on the mother's side is not living. He had trouble with his back, however, which was probably due to an accident. According to information received from his wife, there was little evidence of spondylitis in his case. His wife also stated that neither his father nor his mother, whom she knew very well, had had trouble from arthritis or rheumatism.

Identical twins A and B were separated soon after birth. A has remained in Salt Lake City where the twins were born while B has spent most of his life in New York City. They have met but

once since they were infants. When they were nineteen years of age they spent a few weeks together in Salt Lake City and have not seen each other since. Both boys entered the armed services; one became a glider pilot stationed in the midwest and the other joined the Coast Guard and was stationed in northern California.

Case A

The patient was first admitted to the Salt Lake Veterans Hospital in November, 1948. Ever since he had been on duty in the Coast Guard in 1943, he had experienced a persistent pain in his back. He was hospitalized twice in Naval hospitals and was finally discharged and given a disability because of "nervousness and backache." He followed various occupations including that of fireman on a railroad engine and bookkeeper and laborer for a farm implement company. Six months prior to hospital entry the pain became more intense, persisted longer and involved his entire back including his neck.

Five months before his admission to the hospital he had all of his teeth removed under the impression that it would help his back pain. Two weeks before admission a local physician X-rayed his back and made the diagnosis of spondylitis. He was discharged from his job because of inability to do heavy lifting.

Physical examination revealed tenderness over both sacroiliac joints and pain on lateral flexion and on anterior flexion of the spine. When the patient bent forward, his fingertips reached within six inches of the floor. The chest expansion was 2½ inches. External rotation of the right hip produced pain in the hip but there was no evidence of the disease in any other peripheral joint. The remainder of the physical examination indicated no other abnormalities. The only abnormalities revealed by laboratory tests were an initial WBC of 16,000 which later returned to normal and a sedimentation rate of 17. mm./hr. (Wintrobe).

X-rays of twin A revealed the sacroiliac joints to be hazy with spotty areas of demineralization at the edges. X-rays of the lumbar spine revealed a normal curve with no intervertebral calcifications except for one well-marked syndesmophyte extending between T-12 and L-1. (Figure 1, Plates *A*1 and *A*2.)

The patient has been admitted on three other occasions and has received as much X-ray therapy to the spine as is thought safe. Although at first encouraged by the relief of pain following therapy, the patient later described tenderness over the iliac crest, pain in the symphysis pubis and sternal tenderness and pain when last seen in October, 1949. He has noted more and more that his neck tends to stiffen and that he can do no heavy lifting. No sedimentation rate has ever been above 20, while the great majority have been well within the normal limits. There has never been any evidence of anemia.

Case B

Twin B writes, "In September, 1943, while on active duty as a glider pilot, I awoke with a terrific pain in my left leg. For one week previously I had been having a malaise which weakened me considerably. I was in the hospital for three months with arthritis which struck every joint in my body including the back and hips. There was no swelling of the joints, etc."

For data on this twin we have to depend on correspondence with him and upon the report of Dr. Russell L. Cecil of New York City who very generously supplied us with the data. X-rays taken in 1947 by Dr. Cecil showed partial destruction of both sacroiliac joints. Deep X-ray therapy was begun. He was seen again in November, 1948. At that time he was able to engage in such activities as bowling. He did report some limited motion in the hips and pain in the shoulder joints on elevation of the arms. The sedimentation rate had always been normal (4. mm./hr. Westergren). He had further X-ray therapy in December, 1948 but noted limited relief. By January, 1949, sharp pains had developed in the right hip and in the neck. He was apparently able to continue working but was in moderate, constant discomfort. At no time was his WBC elevated nor was there anemia.

X-rays were obtained in September, 1949, from Dr. Ernest Kraft of New York City. These showed a fuzzy, somewhat irregular contour of the joint space of both sacroiliacs with marginal sclerosis. The lumbar spine appeared free from pathologic change except for sacralization of L-5. Of special interest was incomplete osseous bridging of the intervertebral spaces between T-10, 11, and 12 laterally. (Figure 1, Plates *B*1 and *B*2.)

Nature of Inheritance

The fact that these identical twins, reared apart and under different conditions, should develop spondylitis at almost the same time and in almost identical parts of the spine, strongly suggests an hereditary basis for this defect. A careful examination of the families of both the father and the mother of the twins shows no other cases of spondylitis and no other proved cases of peripheral arthritis. This gives little information from which to determine the pattern of inheritance. There seems to be no evidence of the modifying factors referred to by Neel[4] and by Claussen and Kober[2]. The manifestation of the trait might be

due to a recessive gene or it may even be the result of a dominant mutation. The high incidence of affected males that has been reported suggests the involvement of a sex-linked recessive factor. From the limited data available in this kindred, one can do little more than speculate as to the pattern of its inheritance.

Summary and Conclusions

The physical examinations, X-rays, and laboratory tests revealed identical symptoms of spondylitis in identical twins who had been reared apart from infancy. The onset in both occurred while at different military posts in 1943. Of particular interest is the lack of persistent elevation of sedimentation rates in both cases. Also of importance is the particular type of involvement revealed by X-rays. Definite sacroiliac change and relative freedom of the lumbar spine with syndesmophyte formation at the thoraco-lumbar juncture were present in both twins, (Figure 1*A-B*). Neither twin showed satisfactory response to X-ray therapy and symptoms are progressing in both at the present time.

The fact that identical twins reared apart should develop identical symptoms of spondylitis at approximately the same time is additional evidence of an hereditary basis for this defect. Since a careful study of the family reveals no other cases of the disease, it is impossible to determine with any degree of certainty the nature of the inheritance of spondylitis in this kindred.

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A NEW INSTITUTE OF GENETICS

ESTABLISHMENT of an Institute of Genetics at the Ohio State University has been authorized by the University Administration. Among its activities, the new scientific organization will coordinate current research programs in genetics and will foster new studies in several departments and colleges of the Universtiy. It will also coordinate instructional programs in this science, both at the undergraduate and graduate levels.

The inauguration of the Institute is an outgrowth of many years of research and teaching on the Ohio State campus and at the Ohio Agricultural Experiment Station at Wooster. In a more limited way, it represents a culmination of extensive group interest which dates back to 1934. Since then, a weekly seminar in genetics has been conducted by the Department of Zoology and Entomology which has been attended by faculty members and graduate students from a dozen or more departments of instruction.

Under the enlarged formal program, the seminar in genetics will be continued. The Institute also will sponsor special symposia and lectures.

Chairman of the governing body of the Institute will be Prof. D. C. Rife, of the Department of Zoology and Entomology. Other members of the executive committee include: R. G. Jaap, Poultry Husbandry, vice-chairman; L. C. Ferguson, Bacteriology, secretary-treasurer; L. C. Saboe, Agronomy; and J. N. Spuhler, Sociology.

HETEROCHROMIA IN PEROMYSCUS*

A Study of Mixed Black and Red Eyes in the Deer Mouse

R. R. Huestis and Ruth Willoughby
University of Oregon, Eugene

MOST of us have eyes which are about the same color but most of us have seen, on rather rare occasions, a fellow Homo who had one eye somewhat or even greatly different in color from the other one. In American publications color reproductions of objects of gastronomic or sartorial interest greatly outnumber those devoted to science. But heterochromia did score in *Life*[3] with a full page color reproduction of the face of a symmetrical young woman with the interesting asymmetry of a blue and a brown eye.

Heterochromia has been reported as occurring in various domestic animals[5]. In one residential area of Eugene, where drift or perhaps natural selection produced a high incidence of white cats, the number of white cats with one blue and one yellow eye appeared, to one of us, to approximate the number having both eyes blue or both eyes yellow; a condition of affairs which would make it reasonable to suppose that the asymmetry was inherited and cats prone to consanguinous unions.

Good fortune provided us with the opportunity to test the inheritance of heterochromia in the deer mouse and this paper is a report upon the rearing of 877 young mice, among which 113 had a red eye on the left side and a black one on the right and 116 had a red eye on the right side and a black one on the left. The nearness to equality in these two numbers, together with data from matings designed to test the point, makes it highly probable that, in the deer mouse at least, heterochromia is not accompanied by a tendency toward sinistrality or dextrality as many asymmetries are.

The character which exhibits this asymmetrical deposit of pigment in the eyes of certain proportions of individuals, in the young from different matings, is called red-eye. It was first observed and tested when a single male with both eyes red appeared in a litter of brown silver mice, a pelage color combination that normally has black eyes[2]. The difference in eye color was not accompanied by any observable pelage color difference and this has proved to be the case in all the individuals we have observed since. The difference in expressivity of the character, some mice having both eyes red and some only one, became obvious as soon as new litters containing red-eyed individuals were produced.

Methods

Young mice are examined soon after they are a month old, the age at which they are usually separated from the mother. The examination table has a 200-watt bulb and a reflector. The eyes of some mice are more protuberant than the eyes of others which makes the eye color easier to identify but reasonably accurate classification is not difficult in most cases. We have found that doubtful cases are usually due to some black eyes having pale sectors in them or some red eyes having pigmented sectors in similar positions. A considerable number of eyes has been removed from individuals that were being discarded and these eyes have been examined or dissected under low power magnification or fixed and sectioned.

Description of Red Eye

The eyes of wild-type Peromyscus from the Eugene area are heavily pigmented over the retina, ciliary body and iris. The main branches of the retinal blood vessels divide the rear of the eye of Peromyscus at its equator, into dorsal and ventral hemispheres. A part of these main branches can usually be seen when a fully pigmented eye is examined under a low power binocular. Eyes of brown mice contain less pigment and, under the binocular, the ramifications of the retinal blood vessels show

*This research was assisted by grants from the Graduate Division of the University of Oregon.

up like white tracings on smoked paper.

Red eyes have a sharply defined ring of pigment coincident with the position of the ciliary processes. This is to a considerable extent due to the convolutions of the pigmented epithelium in this region and perhaps to more pigment granules per cell. The iris of red eyes is underlain with a continuous but thinner layer of pigment, looks smoky rather than black, and so contrasts with the ciliary ring. The retina proper of red eyes contains very little pigment and this is the main reason the eye looks red. Under low power magnification the retina of red eyes does not appear to have any pigment at all, the eye glows warmly in light, and if held cornea down it gives sharp images of illuminated objects on the table. Sections of red eyes show, under high power, that the inner epithelium of the retina of red eyes does contain a little pigment. Red eyes do not average as pale as the eyes of ivory, pink eye or albino mice but do overlap ivory and pink eye[4, 1].

All the mice reported upon in Figure 3 are phenotypically brown. We have also reared about 100 young mice in litters in which half the young were brown and half wild-type or silver. Red eyes could be identified in the brown young only. This seems to be due to the fact that wild type or silver mice have an iris too dark to allow a lack of pigment in the retina proper to be seen during the usual inspection. Brown pelage is, however, not essential to the production of red eyes. Non-brown pink-eye mice, which also carry genes for red eye, exhibit heterochromia; the individuals have a paler red eye on one side of the head, a darker red eye on the other. Except for these detectable cases pink-eye, in which the eye color varies directly with the pelage color, is epistatic to red eye. The pink-eye red-eye complex does not have an eye color which can be distinguished from that of light-pelaged pink-eye mice.

A spotted retina, an otherwise black retina proper with a pale sector or an otherwise pale retina with a black sector, rather frequently exhibits a sharp margin to the pigment along one of the main retinal vessels which divide the rear of the eye into dorsal and ventral hemispheres. It looks as though a large blood vessel can act as a barrier to pigment spread although it does not always do so. Pigment deposition in a dark retina tends to be heavier in proximity to the ciliary body and to thin out somewhat near the entrance of the optic nerve.

Results

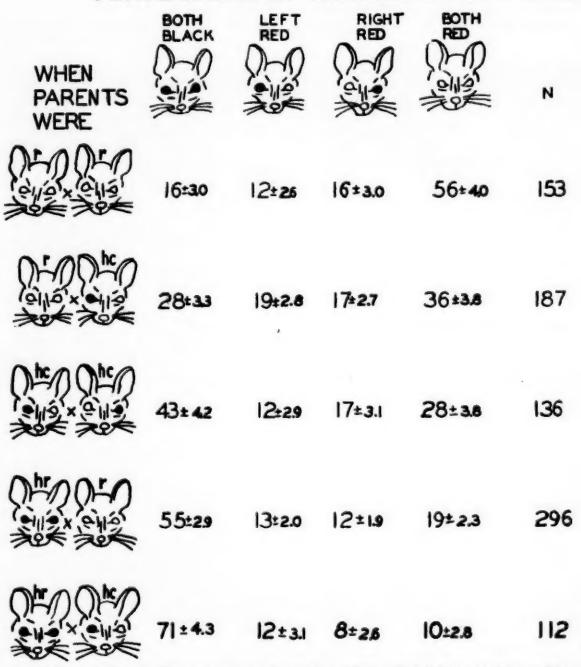
Figure 3 lists the results of our various matings in percentages of young mice with both eyes black, the left red, the right red, or both eyes red. Parents are arranged in terms of increase in eye pigment from top to bottom. All parents had themselves at least one parent with both eyes red. Symbols used are *r* for both eyes red, *hc* for heterochromia and *hr* for a black-eyed mouse with a parent having both eyes red and a parent having both eyes black. The standard errors of these percentages vary between two and five percent. A difference between percentages should be over six to be significant and might not be significant unless it is close to twelve. The general trend is highly significant.

As the total amount of pigment in the parents' eyes increases the percentage of young mice with both eyes black increases directly in a very regular manner. Plotted on coordinate paper these percentages of young black-eyed mice in different matings lie in a straight line. The percentage of mice with heterochromia has its maximum in the offspring of $r \times hc$ parents and thereafter diminishes as the amount of pigment in the parents' eyes increases. The percentage of young mice with both eyes red diminishes as the amount of black in the parents' eyes increases and the diminution is much greater than in the case of the young mice with heterochromia. When parents have four red eyes between them the percentage of *r*'s is twice that of *hc*'s. When parents have but one red eye between them the percentage of *hc* young is twice that of *r* young.

The sex ratio in the various classes of young mice did not deviate significantly from 100 so the percentages have all been computed upon the combined total of both sexes.

Interpretation

We are making the provisional assumption that red-eye is due to a recessive single gene substitution. The first red-eyed mice observed were the offspring of black-eyed parents. Matings of red-eyed mice with stock black-eyed mice usually produce all black-eyed young, and exceptions to this may reasonably be supposed to be due to some spread of the gene for red-eye through-



RESULTS OF MATING RED-EYED AND BLACK-EYED MICE

Figure 3

This pictorial tabulation shows the distribution of monochromic and heterochromic eyes among the progeny of various matings. The symbols used: r = red; hc = heterochromic and hr = heterozygous black-eyed mice with one red-eyed parent. The proportion of black-eyed mice in the progeny is highest when the number of black eyes among the parents is the greatest.

out the colony before it was discovered.

The differences in penetrance and expressivity could be accounted for by the assumption that red-eye acts like a gene for pattern spotting along an axial gradient which is influenced by modifying genes. These might modify the position of eye pigment like the modifiers of the hooded skin pattern in rats which exhibited spot asymmetries as well as differences in the amount of hood. This assumption is merely a thrust into the arras and we await cries of pain. The theory could account for the spotted retinas and might be checked by seeing if the pale spots on a dark retina tended to be posterior and the dark spots on a pale retina anterior in position.

Summary

1. Red-eye in Peromyscus reduces the pigment in the eyes without affecting the pigment of the pelage.

2. The effect may be bilateral and reduce pigment in both eyes equally or reduce pigment in either the right or the left eye in equal proportion of cases. The heterochromia thus produced is comparable to that observable in man and other animals.

3. The percentage of mice with heterochromia in one type of mating equaled the percentage in which a similar amount of pigment reduction had taken place in both eyes.

4. There may be a reduction in pigment in only a portion of one eye. This effect is not obvious under the type of inspection used to identify the classes of mice reported upon here.

5. All the young mice listed in Figure 3 had brown pelage but heterochromia takes place in pink-eye mice which are not genotypically brown.

6. The less pigment present unilaterally or bilaterally in the eyes of parent mice the smaller is the proportion of their young which have both eyes black.

7. Generally speaking the greater the pigment reduction in the eyes of parent mice the greater is the proportion of young mice that have one red eye or both eyes red but the gradient of difference is not the same in these two latter classes of mice.

8. It is tentatively assumed that red-eye is a recessive gene substitution with an effect observable in the pigment of the eye and readily modified by other genes present in the population.

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◆

A Story of Plant Exploration

THIS brief volume* originally presented as a doctoral thesis, undertakes a concise account of the introduction of foreign plants into the United States. Beginning with the Colonial period it goes on to the contributions of such men as Benjamin Franklin, George Washington, and Thomas Jefferson, as well as the role of our Federal Government, first through the Patent Office and later through the Commissioners of Agriculture, the last chapter touching on the Twentieth Century with a final paragraph entitled—"Looking to the future."

The author as a social scientist explains that his aim is to present to the layman—"A historical account of the introduction of America's present crops, to close a gap in the story of American economic development." Any attempt to correlate one science with another is clearly desirable but in leaping from peak to peak one had best be certain of the terrain before taking off.

Space is not available to indicate the numerous errors and defects in this book but it will suffice to indicate them in a general way: failure to number or list the several figures and outline maps; unnecessary inclusion of technical genetical terms; an entirely inadequate index; omission of a glossary to indicate the scientific equivalents of common names of plants; frequent repetitions; and several inexact or misleading statements.

W. Andrew Archer
Bureau of Plant Industry,
Beltsville, Maryland

**America's Crop Heritage—The History of Foreign Plant Introduction by the Federal Government*, by Nelson Klose, The Iowa State College Press, 1950, 156 pp.

◆

The Makro and the Mikro—Or Is It?

(A Letter from Germany)

To the American Genetic Association:

After comparisons succeeded in skulls of homo and sus I could prove a common form for Flora-Fauna representatives—without any publication hitherto. These successes in question I am indebted for my Makrobiogenetischen Analyse further being capable to describe the Creation Story by Makrobiogenet decisions. The cell changing from shrinked tissue into the most extended volume and the climates are in relation—intelligible in Makroskopischen manner.

The acquaintance with the degree of cell's elasticity is as necessary as such chemical parts.

My discipline of the Bewegungsphysiologischen Analyse let recognise already on the object Equus genetische limits with injurius bak effets.

It should be necessary by means of these decisions to spend a new foundation for the nature science universe and indeed more successfull than hitherto the Mikrobiogenetische method could be done alone.

The method of the mathematical report of the chemical-physical processes should be of more clearness in the common way and therefore Makro. method beside Mikro. method.

I am looking for a great foundation in the interest of more clearness for nature science.

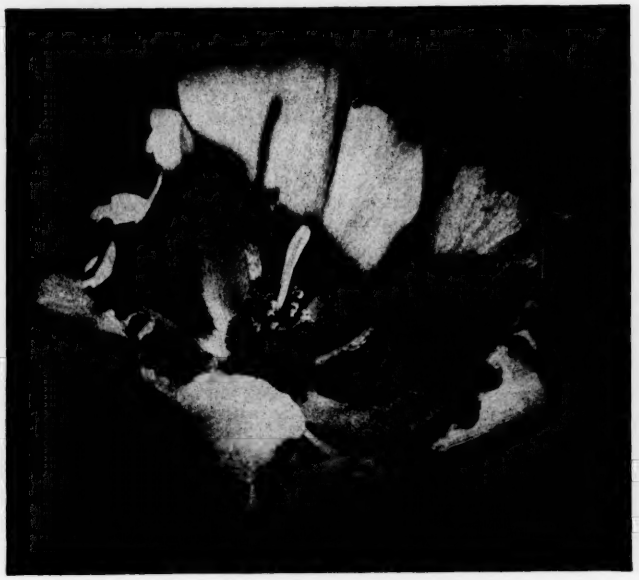
The forms have an intercourse value and therefore a traffic value belonging 5000 skulls sketches saleable and 60000 numbers.

Respectfully,

Hermann Feesche,
Private Learned Man

NEW TRISPECIES HYBRIDS IN COTTON*

META S. BROWN AND MARGARET Y. MENZEL



TRISPECIES FLOWER

Figure 4

This flower is from a plant having chromosome sets from three species of cotton (*hirsutum*, *arboreum*, and *harknessii*). Note the long style and stigma and the large petal spot.

IN the cotton improvement program at the Texas Agricultural Experiment Station, extensive use has been made during the past ten years of selections from a trispecies hybrid combining Asiatic, American wild and American cultivated species of cotton. The original hybrid of *Gossypium arboreum*, $2A_2$,† × *G. thurberi*, $2D_1$, (Asiatic × American wild) has been obtained by several workers, among them Beasley[1, 2] and Harland.[6] After colchicine treatment of the F_1 hybrid, the synthesized amphidiploid, $2(A_2D_1)$, was crossed with the natural amphidiploid *G. hirsutum*, $2(AD)_1$. Successive backcrossing of the trispecies hybrid to *G. hirsutum* has produced strains with a fiber strength considerably above that of our present cultivated varieties.[10]

Other amphidiploids combining a wild species with a cultivated Asiatic species, such as *arboreum-anomalum*, $2(A_2B_1)$,

*Contribution from Department of Agronomy, Cotton Investigations Section, Texas Agricultural Experiment Station, College Station, Texas, as Technical Article No. 1317. A part of the work was done under Project S-1 of the Research and Marketing Act of 1946.

†The genome symbols used in this paper are those devised by Beasley[1]. The letters *A*, *B*, *C*, *D* and *E* designate the genome groups in *Gossypium* which are well differentiated cytologically as well as genetically. Within the diploids, the subscripts, eg. A_1, A_2, etc. indicate genetically distinct species with only slight chromosome differentiation. A second number following a subscript, eg. $D_{2\text{-}1}$,$D_{2\text{-}2}$, indicates species genetically but not cytologically differentiated. For further details see Beasley's paper.

and *arboreum-stocksii*, $2(A_2E_1)$, have been obtained by Beasley.[3]. These two amphidiploids produce sterile trispecies hybrids when crossed with *G. hirsutum*.[4] Considering the chromosome constitution of *G. hirsutum*, $2(AD)_1$, and its putative origin from Asiatic and American wild diploids,[2, 11, 12] these last two groups of species are judged to offer the most promise in a breeding program with the American Upland cottons.

Although the synthesized *arboreum-thurberi* amphidiploid is similar in genome constitution to *G. hirsutum*, crosses between the two behave in F_1, in F_2 and in backcrosses more as hybrids between distinct, though closely related, species than as intervarietal crosses of the same species. The synthetic amphidiploid may be regarded as a new species similar to the three natural amphidiploid species *G. hirsutum*, *G. barbadense* and *G. tomentosum*. For the purpose of a long range breeding program as well as that of further elucidating the precise origin of the natural amphidiploids, it is desirable to obtain and compare other trispecies combinations of *G. hirsutum*, Asiatic and American wild species.

The technique of crossing two diploid species, and then treating the F_1 hybrid with colchicine to produce a fertile amphidiploid with the same chromosome number as *G. hirsutum*, fails in many instances because some American wild species cannot be crossed directly with Asiatic, and others cross only with difficulty. The F_1 hybrid *G. arboreum* × *G. harknessii*, for example, has not been obtained. This difficulty has been overcome recently in this laboratory by crossing hexaploids of *G. hirsutum* and a diploid species with another diploid species.

Six new tetraploid trispecies hybrids have been obtained in this manner. They are: *hirsutum-herbaceum-harknessii*, from the hexaploid *G. hirsutum* × *G. herbaceum*, $2((AD)_1A_1)$ × *G. harknessii*, $2D_{2\text{-}2}$; *hirsutum-arboreum-harknessii*, from the hexaploid *G. hirsutum* × *G. arboreum*, $2((AD)_1A_2)$ × *G. harknessii*; *hirsutum-anomalum-harknessii*, from the hexaploid *G. hirsutum* × *G. anomalum*, $2((AD)_1B_1)$ × *G. harknessii*; *hirsutum-stocksii armourianum* and *hirsutum-stocksii-harknessii*, from the hexaploid *G. hirsutum* × *G. stocksii*, $2((AD)_1E_1)$ × *G. armourianum*, $2D_{2\text{-}1}$, and *G. harknessii*, respectively; and *hirsutum-stocksii-raimondii*, from

TABLE I.—Meiotic pairing in the *hirsutum-arboreum-thurberi* trispecies hybrid, $A_2D_1(AD)_1$.

II	III	IV	V	VI	VII	VIII	X	I	No. PMC
16	1	3	1						1
16	1	2				1		1	1
16				3				2	1
17	2	2						4	1
18		4							1
18	2		2						1
18		**2**		**1**				2	1
18	3	1						3	1
18		1				1		4	1
19	2	2							1
19		2		1					1
19	1	1		1				1	3
19		1				1		2	1
19	1	2						3	1
20	1	1	1						1
20	4								1
20		3							2
20		1				1			1
20	2	1						2	1
20		2						4	1
21	2	1							3
21		1		1					2
21							1		1
21	3							1	1
21		1	1					1	1
21	1		1					2	1
21		2						2	1
22	1		1						1
22		2							3
22						1			1
22					1			1	1
22				1				2	1
22		1						4	2
23	2								1
23				1					5
23			1					1	1
23		1						2	1
24		1							6
24	1							1	1
26									1
Total 1214	36	59	8	16	1	5	1	51	58
Avg. 20.93	0.62	1.01	0.14	0.27	0.02	0.08	0.02	0.88	

Average number of chromosomes paired: 51.11

TABLE II.—Meiotic pairing in the *hirsutum-arboreum-harknessii* trispecies hybrid, $(AD)_1A_1D_{2\text{-}2}$.

II	III	IV	V	VI	I	No. PMC
18	3	1			3	1
19	2			1	2	1
19		3			2	1
19	2	1			4	1
19		2			6	1
20		3				2
20		1		1	2	1
20		2			4	3
21		1		1		4
21		2			2	6
21	1	1			3	1
22		2				7
22	1	1			1	5
22		1			4	2
23			1		1	1
23		1			2	3
23	1				3	1
24		1				12
24	1				1	7
24					4	1
25					2	6
Total 1498	21	73	1	6	92	67
Avg. 22.36	0.31	1.09	0.01	0.08	1.37	

Average number of chromosomes paired: 50.63

the hexaploid *hirsutum-stocksii* × *G. raimondii*, $2D_5$.

Cytology and Morphology

The cytological behavior at meiotic metaphase in the *arboreum-thurberi-hirsutum* hybrid has been reported by Beasley.[2, 3] He found usually 24-25 bivalents at metaphase I, with one or more multivalents in most cells, and one or more univalents in about one-third of the cells. Current cytological data for the same hybrid are given in Table I. Usually one or more multivalents are present, with a maximum of five; as many as ten chromosomes may be associated in one multivalent.

Cytological data for the two new hybrids are summarized in Tables II and III. In the *arboreum-harknessii* hybrid, an average of 50.6 of the 52 chromosomes pair at metaphase. The number of bivalents ranges from 18 to 25, with 24 the most frequent number. One or more multivalents, with a maximum of four, may be present, most often as quadrivalents, rarely as pentavalents or hexavalents. The average number of univalents is 1.37. In the *herbaceum-harknessii* hybrid, the average number of chromosomes paired is 50.16, the average number of univalents 1.84. As in the *arboreum-harknessii* hybrid, one or more multivalents, with a maximum of six, may be present; these are most often trivalents and quadrivalents, with an occasional hexavalent.

The main difference between the two *harknessii* hybrids lies in the frequency with which different multivalent types are found; these are predominantly trivalents in the *herbaceum* hybrid and quadrivalents in the *arboreum* hybrid.

Pairing in the three trispecies hybrids is compared in Table IV. The two hybrids involving *harknessii* are more similar to each other than to the *thurberi* hybrid, the principal difference lying in the maximum number of chromosomes associated in one multivalent (six in the former, ten in the latter). The *thurberi* hybrid also averages a slightly higher number of chromosomes per cell paired as multivalents, as can be seen from its slightly lower average number of bivalents and univalents, and slightly higher average of total chromosomes paired. So far as chromosome pairing is concerned, the fertility of the new hybrids should equal that of the *thurberi* hybrid.

In the new hybrids the characters of *G. harknessii* are quite evident. Stem, leaves and other plant parts are glabrous. The leaves are smooth and dark green, and intermediate in size between those of *G. hirsutum* and the small, slightly dissected leaves of *G. harknessii*. The bracteoles, which are small and caducous in *harknessii*, in the hybrids resemble those of *G. hirsutum* in shape and number of teeth, but are much smaller. The flower of the *arboreum* hybrid is large (Figure 4), slightly ruffled, bright yellow, with a conspicuous petal spot, red filaments, deep yellow pollen, and long style. The *herbaceum* hybrid has smaller, lighter yellow, non-ruffled flowers.

In the *arboreum* hybrid, pollen is shed abundantly; in the *herbaceum* hybrid, pollen is shed poorly and infrequently. The length of the style may interfere with natural selfing in either hybrid, since the stigma extends far

TABLE III.—Meiotic pairing in the *hirsutum-herbaceum-harknessii* trispecies hybrid, $(AD)_1A_2D_{2-2}$.

II	III	IV	VI	I	No. PMC
14	4	2		4	1
19	2		1	2	1
19		3		2	1
20	4				3
20	1	2		1	2
21	2	1			3
21	3			1	1
21		2		2	1
22	1	1		1	4
22	2			2	1
22			1	2	1
22		1		4	1
23	2				1
23	1			3	4
23				6	1
24		1			1
24	1			1	1
24				4	3
25				2	6
Total 819	42	20	2	68	37
Avg. 22.14	1.14	0.54	0.05	1.84	
Average number of chromosomes paired: 50.16					

TABLE IV.—Comparison of chromosome pairing in three trispecies hybrids.

Genome	II	III	IV	V	VI	VII	VIII	X	I	Av. paired	Maximum II	Maximum I	Maximum in multivalent association
$A_2D_1(AD)_1$	20.93	0.62	1.01	0.14	0.27	0.02	0.08	0.02	0.88	51.11	26	4	19
$(AD)_1A_2D_{2-2}$	22.36	0.31	1.09	0.01	0.08				1.37	50.63	25	6	12
$(AD)_1A_1D_{2-2}$	22.14	1.14	0.54		0.05				1.84	50.16	25	6	20*

*In one exceptional plate. The next highest number of chromosomes paired in multivalents, seen in several cells, was 12, consistent with the $(AD)_1A_2D_{2-2}$ hybrid.

beyond the staminal column, as in *G. harknessii*. However, reciprocal backcrosses with *G. hirsutum* have been made with both hybrids. Backcross plants are now being grown for further backcrossing and selfing.

In the remaining new trispecies hybrids, *hirsutum-anomalum-harknessii, hirsutum-stocksii-armourianum, hirsutum-stocksii-harknessii,* and *hirsutum-stocksii-raimondii,* the Asiatic genome has been replaced by the genome of a second wild species. A fertility equal to that of the *arboreum-thurberi* or *arboreum-harknessii* hybrids is therefore scarcely to be expected. Comparative tests are now in progress. The hybrids are of great cytological interest because each of them involves two diploid species which have not been crossed directly. The comparative cytology of these hybrids will be described later in relation with other hybrids involving the *anomalum, stocksii,* and American wild genomes.

Discussion

Beasley,[1] Skovsted[12] and others, including the authors, have studied the cytological behavior in F_1 hybrids of $A_2 \times D_1$, $(AD)_1 \times A_1$ or A_2 and $(AD)_1 \times D_1$ or D_2. Some pairing does occur between chromosomes of the A and D genomes in F_1 hybrids of $A_2 \times D_1$; but in combinations containing three or more complete genomes, as in F_1 hybrids of $(AD)_1 \times A$ or D species, pairing is preferential within the A or D genomes. These relationships are discussed further by Brown (in press). A detailed study of chromosome pairing is now in progress in a series of hybrids involving A_1, A_2, $(AD)_1$ and several members of the D group. This comparative study is designed to show to what extent each of the genomes is responsible for certain cytological configurations in complex hybrids, including trispecies tetraploids, which involve different A and D genomes.

Iyengar[7] reported a fertile tetraploid of the constitution $(AD)_1A_1D_{2\text{-}1}$ from a cross of the hexaploid *G. hirsutum* × *G. herbaceum* by *G. armourianum*. At meiosis many cells had 26 bivalents, with a mean value of 24.3 bivalents, 2.35 univalents and 0.35 trivalents. An earlier report by Mauer[9] of a trispecies hybrid which gave 26 bivalents at meiosis is somewhat doubtful. A highly fertile tetraploid which closely resembled *G. barbadense* was obtained from a cross of the F_1 hybrid *G. barbadense* × *G. thurberi* by *G. arboreum*. The absence of any mention of multivalents or univalents such as were found in the three hybrids described above suggests that Mauer's tetraploid may not have contained the *arboreum* and *thurberi* genomes.

Although the actual amphidiploid of *arboreum-harknessii* has not been obtained directly, the trispecies hybrid resulting from the hexaploid × diploid method of crossing is equivalent to that obtained by crossing the *arboreum-thurberi* amphidiploid with *G. hirsutum*. A comparison of the three trispecies hybrids allows us to deduce that the *arboreum-harknessii* amphidiploid, if it could be obtained, would be as closely related to the natural amphidiploid species as is the *arboreum-thurberi* amphidiploid. If anything, the *harknessii* hybrids show less complex pairing relations than the *thurberi* hybrid. The *D* genome of *hirsutum* does not appear to be completely homologous with either *G. thurberi* or *G. harknessii,* and these two species are *cytologically* about equally acceptable as the putative American wild parents of *G. hirsutum*.

The character from *G. thurberi* on which most emphasis has been placed in the current breeding program is that of high fiber strength. Another character of potential value which has been reported for this species is resistance to pink boll worm[8]. The species is known to be frost resistant under natural conditions, and has shown itself to be hardy and prolific under both greenhouse and field conditions.

The characters of *G. harknessii* which might be of value if transferred into commercial cottons include the smooth, shallowly lobed leaf, and the small, caducous bracteole, both of which would facilitate the production of cleaner seed cotton. There is evidence also that the waxy covering on the seed hairs of *G. harknessii* is different from that of *G. hirsutum,* and may contribute to the luster and special spinning properties of the lint of selected backcross lines. The

semi-arid habitat of *G. harknessii,* which is native to Lower California, indicates that drought tolerance, also, is present in this species. The unforeseen recovery of high fiber strength among the backcross segregates of the trispecies hybrid of *G. thurberi,* which is a practically fiberless species, suggests that with careful selection, other as yet undetermined characters may be recovered from the *harknessii* hybrids. In addition, characters known to be present in the Asiatic parent, *G. arboreum,* are subject to selection. Among these is resistance to bacterial blight, known also as angular leaf spot, boll rot, or blackarm.

Summary

Two tetraploid trispecies hybrids produced by crossing the hexaploids of *G. hirsutum* × *G. herbaceum,* and of *G. hirsutum* × *G. arboreum* by *G. harknessii* are described. Chromosome pairing is compared between the two hybrids and with the *arboreum-thurberi-hirsutum* trispecies hybrid. Four additional new trispecies hybrids, each involving two wild species, are reported. The six new hybrids each involve two diploid species which cannot be crossed directly.

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STATISTICAL ANALYSIS IN BIOLOGY

A Review

THE second edition of this book* is identical page for page with the first edition, except for the correction of some misprints and errors which occurred in the previous edition, and the thirteenth chapter which is entirely new.

The class of reader for which this book was prepared is specified by the title. The author has chosen biological examples, so that only the language and symbols of statistics will be new to his readers. He has deliberately selected those statistical methods which are appropriate to many of the more important problems in the analysis and interpretation of a wide variety of situations in biological experimentation. The examples he has chosen are used to present, step by step, the arithmetic and logical processes needed to reduce the data from an experiment and to interpret the results. By showing the relation of the analysis and interpretation to the design in each experiment used as an example, the author has presented many of the essentials of good experimental design.

The reviewer is especially pleased by the presentation of the interrelation of the normal, the t, the Chi-Square, and the z distributions and their uses in statistical analysis. It would seem, however, that it would be more precise to speak of the distribution of the variance ratio than of z in this connection. The emphasis on individual comparisons or degrees of freedom and the principles of partition is well placed.

Though a number of corrections and changes have been made in preparing the second edi-

**Statistical Analysis in Biology,* Second Edition, American Photo Offset Reprint, K. Mather (Head of Genetics Department, John Innes Horticultural Institute, London), New York 3, Interscience Publishers, Inc. 1947. Pp. 267. $5.00.

tion, such as the mean square for joint regression in Table 28, a number of other points might well receive consideration when the book is again revised in preparation for the third edition, such as the following:

1. The wisdom and necessity of the statement, "a χ^2 is seldom obtained directly as the ratio of two variances or sums of squares."
2. Instead of saying, "Each total is composed of 12 values, and so after squaring and summing the results must be reduced to 1/12," it would seem that he should say, ". . . must be divided by 12" or ". . . must be multiplied by 1/12."
3. Ways of determining the various sums of squares in cases of several criteria of classification that are less cumbersome than those given in the next to the last paragraph on page 76 are in common use.
4. The use of the expression "incomplete analysis" on pages 79, 129 and 170 seems unnecesary and unfortunate. The reviewer has been asked by several persons to whom he had recommended the book, just how such analyses could be completed.
5. The typographical effect of using a bar over the Greek letter Xi on page 142 to indicate the mean value of Xi is somewhat confusing.
6. The word "of" after the word "deviation" in the first sentence following Table 45 on page 191 should be deleted.
7. The expression "Applying these three tests to the partition of a 2 degree of freedom into two simple components . . ." would be more readily understood if 'sum of squares' or equivalent expression were inserted between the words freedom and into.
8. In presenting the calculation of a heterogeneity χ^2 in a $2 \times j$ table, the author handles subscripts and symbols for summation in a manner that is below the standard of the book as a whole. He might improve this treatment by saying, "Each line of the table contributes an item to the total χ^2, that of line 1 being

$$\frac{(m_2a_{11} - m_1a_{12})^2}{m_1m_2n_1}$$

and by using j as the first element in the subscript of a and as the subscript of n in each expression preceded by a summation sign as

$$\frac{S\,[(m_2a_{j1} - m_1a_{j2})^2/n_j]}{m_1m_2}$$

Chapter XIII on transformations is a praiseworthy addition to a good book. It is unfortunate, however, that the author, like many of those who have published on transformations, has selected an example to illustrate the value of the angular transformation in which the conclusions to be drawn using the t-test are the same whether the angular transformation is used or not. Ignoring the author's statement, "Similarly, we are debarred from the use of such a simple and powerful technique as the analysis of variance . . . ", the reviewer applied the analysis of variance to the proportions given by the author in Table 59. For comparison, the values of t and P secured with and without the transformation are given in the following table using the author's identifications of the various items:

Item	D/F	With Transformation		Without Transformation	
		t	P	t	P
General Deviation	1	2.491	0.02–0.01	2.474	0.02–0.01
Difference of W and R means	1	1.232	0.3–0.2	1.168	0.3–0.2
White series, Deviation	1	1.123	0.3–0.2	1.091	0.3–0.2
Red series, Deviation	1	2.245	0.05–0.02	2.273	0.05–0.02

A reader of this book, whether a biologist or not, who is interested in the techniques which the author has so simply and clearly presented should master them easily and quickly.

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ICHTHYOSIS, A NEW RECESSIVE MUTANT

In the House Mouse

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A NUMBER of mutants are known in *Mus musculus* which affect the coat or skin or both. Most of them fall into one of two main groups, the hair-waving and the hypotrichosis mutants respectively. Grüneberg[3] has summarized previous work on them. Descriptions of three more have subsequently become available, namely *wellhaarig*[4] a recessive hair-waving gene; *fuzzy*[5] a recessive gene which causes the coat to be sparse and curly; and *crinkled*[2] a recessive gene with manifold effects on the hair follicles and skin.

During 1948 an inherited ichthyotic condition was found in a sib-mated stock* maintained at Edinburgh; it occurred in two of several parallel sublines, all derived from three foundation animals. It presumably arose by spontaneous mutation in a common ancestor.

Description

The vibrissae of a two-day-old ichthyotic mouse are straight but they appear to be somewhat shorter than those of its normal sibs. At three days the vibrissae begin to lose their regular arrangement; there is a certain amount of criss-crossing, especially in the region nearest the tip of the nose, and some of them are also curved. At four days most (but not all) of the vibrissae are seen to curve upwards and inwards towards the tips of the nose; dorsal guard-hairs are scarcely visible, though they are clearly visible in the normal mouse of the same age. At six days the dorsal guard-hairs of an ichthyotic mouse are still scarcely visible, being represented only by a little fluffy down on the head and neck; its skin looks rough and papillate, and a number of creases, like those of the palm of a hand, are clearly visible in the skin of its sides, back and tail.

During the second week, when a normal mouse grows a full coat, an ichthyotic rarely grows more than a thin, short coat, in which the density of hairs appears to be low and the hairs themselves curly; the vibrissae are

YOUNG ICHTHYOTIC
Figure 5

During the third and fourth weeks the skin of the ichthyotic mouse becomes hard and scaly and in a few days cracks and is finally sloughed. This mouse has scaly plates of old skin on the nose, ears, legs and tail.

*The authors wish to thank Miss Catherine Scott, who found the first ichthyotic mice, and Miss Esme Mavor for their technical assistance and care in maintaining the stock.

short and heavily curled. The ichthyotic grows more slowly than its normal sibs and is less viable.

During the third and fourth weeks an ichthyotic mouse usually falls far behind its sibs and undergoes a developmental crisis. The skin dries, hardens and becomes scaly; a few days later it cracks and is finally sloughed. There is considerable variation between individuals in the severity of this process. In mild cases the scales are small, confined chiefly to the back, legs and tail and are shed without apparent difficulty. In severe cases the scales form rigid plates, covering the whole body, which appear to make respiration and movement of the limbs difficult. On the tail the dry skin often forms a number of rings; these presumably interfere with the circulation to more distal parts, since necrosis of the tip of the tail is an almost invariable feature of the condition. The sloughing does not occur all over the body simultaneously, but proceeds in an anteroposterior and dorsoventral direction. However, odd scales may remain attached to the nose, ears and legs after the process is completed in neighboring regions. Viability is low during the sloughing process.

An older ichthyotic mouse often grows a thin, curly coat, which never becomes as thick as in a normal mouse; at the most it resembles the coat of a *fuzzy* mouse[8]; at the least the ichthyotic is competely bare except for a few short, heavily curled vibrissae. Ichthyotics usually remain small and are often subject to respiratory disorders. Some are mute, but it is not known whether this is true of most or all of them. The eyelids may be thickened, the lower lip retracted and the claws may be long and spiralled, but these are not regular features.

Most ichthyotics are sterile. An external vaginal opening appears in the female, but it is usually displaced forwards, over the pubic symphysis. The skin between the vagina and anus is then stretched tightly over the posterior edge of the pelvic girdle; the vaginal canal, pinched between the skin and symphysis, is thereby effectively closed. A few females are fertile and they can suckle their litters, (though poorly) but their breeding lives are short and none has yet borne more than two litters. Most male ichthyotics fail to develop a scrotum and the penis is usually deflected forwards; cryptorchid males are invariably sterile, but males with one or both testes descended are usually fertile.

There is some variability in the severity of the ichthyotic condition, but the range of expression does not approach the normal condition. There is never any doubt whether a mouse should be classified as ichthyotic or not.

Inheritance of Ichthyosis

No ichthyotics have yet bred when mated *inter se*, even when both mates had previously been proved fertile.

Matings between ichthyotics and unrelated mice of either sex produced only non-ichthyotic young, thereby establishing that the condition is not inherited as an autosomal dominant, nor is it due to a mutant gene in the differential segment of the sex-chromosome.

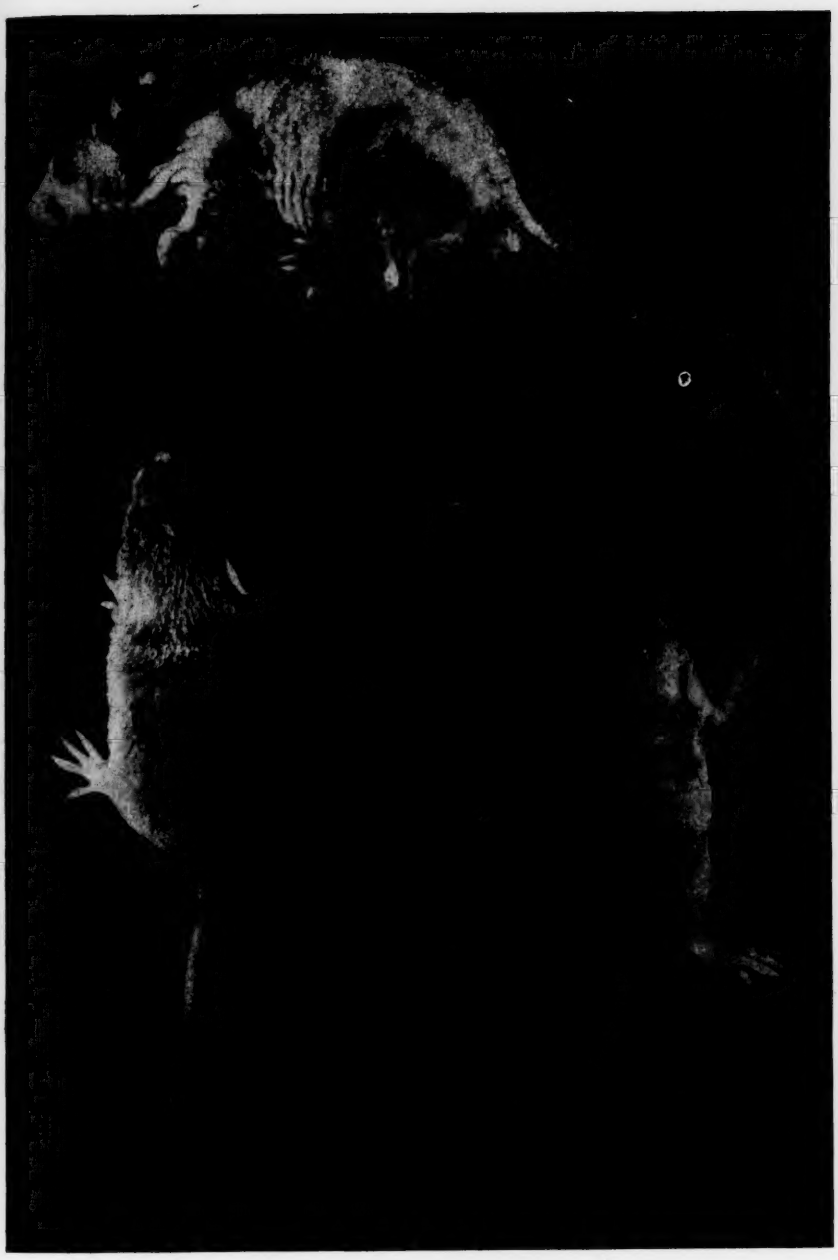
Ichthyotics were recovered in the F_2 and backcross generations, but in numbers which were significantly lower than expectation on the basis of a single Mendelian recessive. The data, nevertheless, are held to support a one-gene hypothesis and the symbol *ic* is proposed for the mutant gene. The deficiency of ichthyotics is attributed to high early

TABLE I.—Segretation of *ic*.

PARENTS	PROGENY									SEGREGATION	
	Classified						Unclassified			Ichthyotics	born
Type of mating	Lived			Died			Died			Calc-	Ex-
	T	N	I	T	N	I	T	N*	I*	ulated	pected
Intercrosses:—											
F_1 ♂ × F_1 ♀ (*Ca* not present)	66	50	16	—	—	—	2	?	?	16.00	16.50
F_1 ♂ × F_1 ♀ (*Ca* present)	415	363	52	55	11	44	32	6.4	25.6	121.60	125.50
Normal ♂ × normal related ♀ (*Ca* present)	147	116	31	16	12	4	14	10.5	3.5	38.50	44.25
Backcrosses:—											
Ichthyotic ♂ × F_1 ♀ (*Ca* present)	12	9	3	—	—	—	—	—	—	3.00	6.00
Outcrosses:—											
Ichthyotic ♂ × normal ♀	43	43	—	—	—	—	—	—	—	—	—
Normal ♂ × ichthyotic ♀	11	11	—	—	—	—	—	—	—	—	—
Hairless ♂ × ichthyotic ♀	5	5	—	—	—	—	1	—	—	—	—
Ichthyotic ♂ × hairless ♀	12	12	—	1	1	—	—	—	—	—	—
Ichthyotic ♂ × fuzzy ♀	6	6	—	—	—	—	—	—	—	—	—

T = total; N = normal; I = ichthyotic.

*The figures in the columns marked with an asterisk were obtained by dividing the number in the 'unclassified, died, total' column in the ratio of the numbers in the 'classified, died, normal' and 'classified, died, ichthyotic' columns. Ten complete litters, totalling 28 young, all of which were unclassified and disappeared within five days of birth, have been excluded from the data.



DEVELOPMENT OF ICHTHYOTIC MICE

Figure 6

A shows an adult male with typical very short thin coat and tail shortened by necrosis of the tip. *B* shows a normal mouse fifteen days old and *C* shows an ichthyotic littermate. The coat of the latter is shorter, but skin lesions have not yet developed.

postnatal mortality of homozygotes, for the following reasons. The strain in which the first ichthyotics occurred was homozygous for the dominant hair-waving mutant *Caracul*[1]; consequently classification for ichthyosis of the original F_2 generation, which provided the greater part of the data, could not be based on the appearance of the vibrissae at three days, but had to be delayed until seven to ten days, when the coat abnormalities are clearly visible. A number of young died before the age of classification; more died between the ages of classification and weaning, and among these the number of ichthyotics was disproportionately high. If two plausible assumptions are made, namely (1) that when a complete litter died before classification, death was due to poor maternal care and did not depend on the genotype of the young, and (2) that the proportion of ichthyotics among the remaining pre-classification deaths was the same as that among the post-classification deaths, then an estimate may be made of the number of ichthyotic young born. This is found to be in good agreement with Mendelian expectation (Table I).

The *ic* gene has been proved to be non-allelomorphic with three other mutants which affect the coat, namely *fz*, *hr* and *Ca*. Non-allelomorphism of *ic* with *fz* and *hr* was proved by direct test; both crosses gave an all-normal F_1 generation. Non-allelomorphism of *ic* with *Ca* is established by the fact that segregation of *ic* and *Ca* occurs in F_1 mice who received both mutants from the same parent.

Summary

Ichthyosis, *ic*, is a recessive mutant in *Mus musculus* which affects the skin and its derivatives. It was found in a laboratory stock at Edinburgh in 1948, having presumably arisen by spontaneous mutation. An *icic* homozygote is distinguishable at three to four days by its curly vibrissae; the first coat is very short and sparse and the individual hairs are curly. During the third and fourth weeks the skin may harden, dry and be sloughed and the distal part of the tail becomes necrotic; the adult may grow a thin, curly coat. Other effects of the mutant may include thickening of the eyelids, retraction of the lower lip, mutism, overgrowth of the claws and cryptorchism. Cryptorchid males and most females are sterile. Viability is impaired. *ic* is not allelomorphic with *fz*, *hr* or *Ca*.

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Tomato Genetics Cooperative

A cooperative is being organized for the purpose of exchanging stocks and information relating to Tomato Genetics. Any workers interested in joining the group should contact C. M. Rick, University of California, Davis, California.

COMPUTING INBREEDING AND RELATIONSHIP COEFFICIENTS

From Punched Cards*

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THE genetic analysis of populations frequently requires computing inbreeding and relationship coefficients from extended pedigrees for groups of individuals. This may be needed for periodic summaries, for planning experimental matings, or for making special studies of accumulated data.

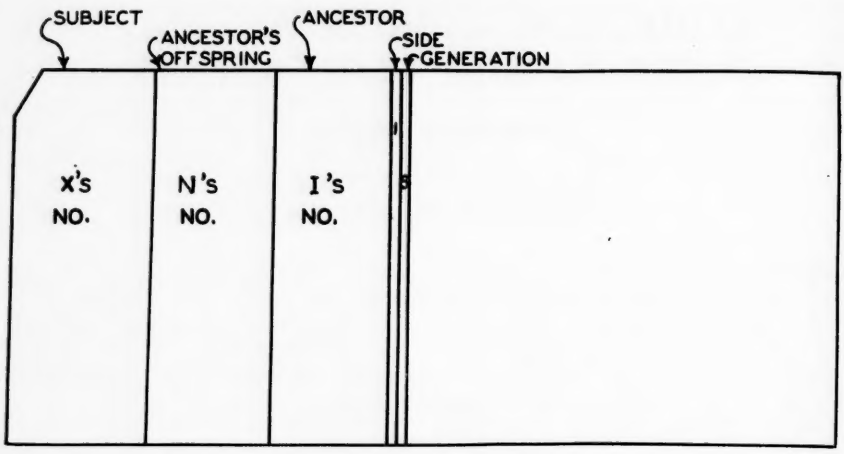
Lush and co-workers[1,3] adapted the punched card system to a sampling process for the pedigree analysis of pure breeds of livestock. The present paper shows how punched cards can be used to reduce the computing of inbreeding and relationship coefficients from complete pedigrees to a routine procedure, largely mechanical and clerical in operation. The method is accurate, even in cases where irregular inbreeding has been practiced, and it saves labor even when the populations being studied are small.

Outline of Method

Briefly the method consists of punching an I.B.M. or similar card for each ancestor of the individuals under study. The numbers or names of the "ancestor," the "ancestor's offspring," and the "subject" are punched on the cards. The number of generations from subject to ancestor and whether the ancestor is reached through the sire or dam of the subject are also punched. Figure 7 indicates how such a card would be punched for the paternal grandsire of the sire of X, whose complete pedigree is shown in Figure 8. One card is punched for the individual itself, which is listed in all three places as subject, ancestor and ancestor's offspring. On the cards for sire and dam the subject is listed both as subject and as ancestor's offspring. On all the rest of the cards the three are different individuals. The cards are machine sorted into hereditary groups ordered numerically by ancestor number, or alphabetically by ancestor name if ancestors are not numbered. The subgroups under each ancestor are ordered by ancestor's offspring. Within each subgroup the cards are in order by subject number. The cards are then tabulated on the usual long folded sheet of paper. This gives a printed list of the data on the cards in the order desired. Table I shows such a list as it would appear for the pedigrees shown in Figure 8. The primary sort was on the column headed "d," the secondary sort was on "c" and the tertiary was on "b." Controls on the tabulating machine are set to single space as long as "c" and "d" remain the same, but to double space when "c" changes, and to double space and also print an asterisk opposite the first card when "d" changes. Controls can also be set to print a "card count" column for each subgroup or for each ancestor.

The numerator of Wright's coefficient of relationship[4], hereafter referred to as the genic covariance, is $\sum [(\frac{1}{2})^{n+n'}(1+F_A)]$, where n and n′ are defined as the numbers of generations from the two subjects to a common ancestor A, and F_A is the inbreeding coefficient of A. The cofficient of inbreeding for X is simply ½ of the genic covariance between the sire and dam of X. This is more conveniently written here as $F_X = 2\sum [(\frac{1}{2})^{n+n'}(1+F_A)]$ where n and n′ are defined as the numbers of generations from subject X through its sire and dam, respectively, to a common ancestor A. The numerical results are identical with those of

*Journal paper No. J-1706 of the Iowa Agricultural Experiment Station, Project No. 1053.



SAMPLE PUNCH CARD

Figure 7

For every ancestor a card coded in this manner is punched giving information regarding every ancestor.

Wright's original formula, the change in definition of n and n′ here being solely a convenience which permits the same cards to be used for computing either the inbreeding of X or the relationship of X to some other individual.

The values for n + n′ are obtained merely by adding the printed numbers for pairs of subjects which are in different subgroups within each ancestor group. For example, the second and third cards listed in Table I yield 1 + 0 for the value of n + n′ as the relationship between Z and Y through the common ancestor Y. But the sixth and seventh cards yield nothing more since they are in the same subgroup; i.e. whatever genes they may have received from R came to both of them thru Y and this relationship has already been counted in the prior listing of Y as a common ancestor.

These values of n + n′ are written in a two-way table, such as Table II, as they occur on the list. After completing Table II, the coefficients for each combination of subjects are reduced by combining powers of ½ to their smallest terms and are then converted to inbreeding coefficients and genic covariances, as shown in Table III. The effect of inbreeding in a common ancestor is included automatically. Only in cases where a common ancestor itself has an inbred ancestor is any additional adjustment needed.

TABLE I.—List printed from punched cards after the cards are sorted into hereditary groups and subgroups.

Page 1 abcdeg	Page 2 abcdeg	Page 3 abcdeg	Page 4 abcdeg	Page 5 abcdeg
*ZZZ 0	*ZSNm2	*ZOIf3	*ZKDf5	*ZNBm3
		YOIf2	ZKDm4	ZNBf4
*ZZYf1	ZRNf3		YKDm4	YNBm3
	YRNm2	ZNIm3	XKDm4	XNBm3
YYY 0	XRNm2	ZNIf4	XKDf4	XNBf3
		YNIm3		
*XXX 0	XQNf2	XNIm3	ZJDf4	ZIBm4
		XNIf3	YJDf3	ZIBf4
*ZZSm1	*XQMf2			ZIBf5
		*XMHf3	*ZKCm4	YIBm4
*ZYRf2	*ZPLm3		ZKCf5	YIBf3
YYRm1	ZPLf4	*ZLGm4	YKCm4	XIBm4
	YPLm3	ZLGf5	XKCm4	XIBf4
XXRm1	XPLm3	YLGm4	XKCf4	
		XLGm4		XHBf4
*XXQf1	*XMKf3		ZICm4	
		*ZLFm4	ZICf4	
*ZSPm2	ZPKf4	ZLFf5	ZICf5	
	ZPKm3	YLFm4	YICm4	
ZRPf3	YPKm3	XLFm4	YICf3	
YRPm2	XPKm3		XICm4	
XRPm2		*ZJEf4	XICf4	
	*ZOJf3	YJEf3		
*ZYOf2	ZOJf2		XHCf4	
YYOf1				

(a) The asterisk is not punched on cards but is printed by the tabulating machine when there is a change of ancestors.

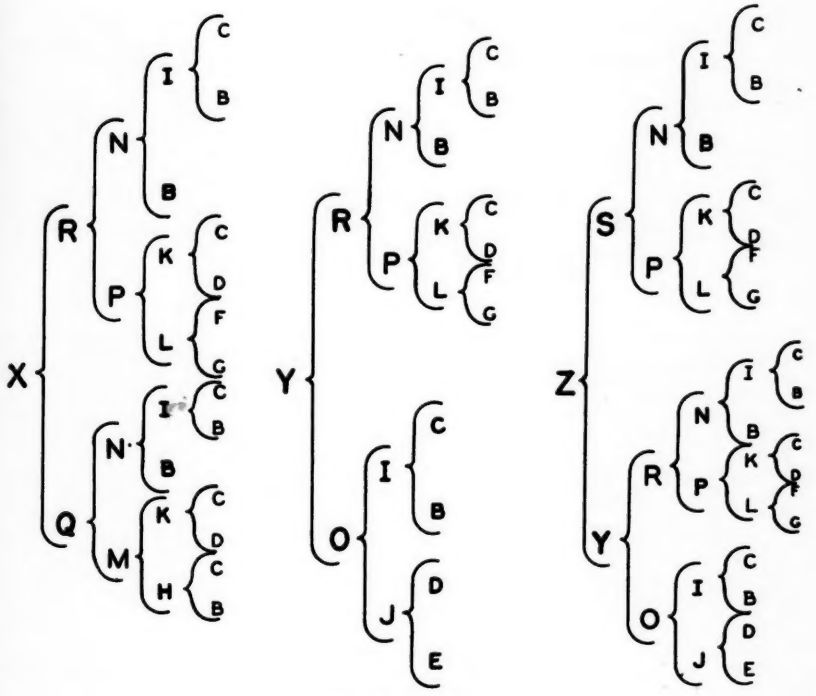
(b) The name or number of subject. It will usually occupy the space of several digits on the punched cards.

(c) The name or number of ancestor's offspring.

(d) The name or number of ancestor.

(e) Sex of parent of subject through which the path is traced. It can be indicated by punching 1 or 2 on punched cards.

(g) Number of generations from ancestor to subject.



SAMPLE PEDIGREES

Figure 8

Here are given three pedigrees of inbred individuals to illustrate the punch card method.

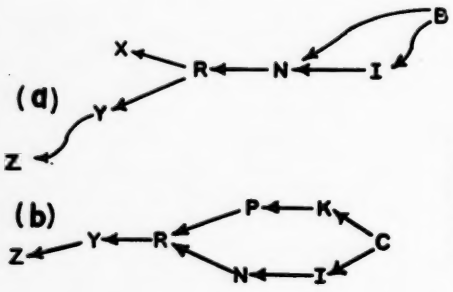
Example

Figure 8 shows the pedigrees of the three individuals, X, Y, and Z which are used to illustrate the method. The punching of the cards from pedigrees as shown there proceeds as follows: XXX O, XXRm1, XRNm2, XRPm2, etc., the final card on the male side of X being XLGm4. The first card on the female side of X is punched XXQf1 and the last one XHBf4. The procedure is the same throughout. After ordering the cards with the sorting machine with the primary grouping on "ancestor," the secondary grouping on "ancestor's offspring," and the tertiary grouping on "subject," the printed list will appear in the order shown in Table I.

The coefficients, $n + n'$, are computed from Table I by adding the number of generations from two subjects to a common ancestor. The first relationship between Z and Y in Table I, shown by the two cards, ZZYf1 and YYY O, has already been mentioned. The $1 + 0$ for $n + n'$ which it contributes is entered in Table II in the cell where the row for Z and the column for Y coincide.

TABLE II.—Values of $n + n'$ computed from printed list.

Subject	$n + n'$ from Table I	Subject Z	Subject Y	Subject X
Z	Page 1	5	14	34
	2	5	4	44576
	3	6	656	66
	4	8899	8788798859	88888999889889889
	5	788	7687778	777888778778889
Y	1			2
	2			46
	3		5	55
	4		77	77888878787
	5		6	777767687
X	1			
	2			46
	3			
	4			8888
	5			7778



INBREEDING AMONG COMMON ANCESTORS

Figure 9

Diagram from pedigree in Figure 8. The upper diagram (*a*) shows *N* as an inbred ancestor of *R* and a common ancestor of *X*, *Y*, and *Z*. Diagram *b* shows *R* as an inbred ancestor of *Y* and a common ancestor of *Y* and *Z*. The value for $n + n'$ computed by the punch card system must be adjusted for such cases.

The three cards ZYRf2, YYRm1, and XXRm1, show relationships for ZX and YX, but none for ZY since the cards ZYRf2 and YYRm1 are in the same subgroup. Their being in the same subgroup shows that they both trace to R through the one gamete which R gave to Y. Therefore the relationship here between Z and Y will already have been counted under Y as an ancestor. These two values of $n + n'$ for relationship through R are 3 for the ZX cell and 2 for the YX cell in Table II.

The first evidence of inbreeding is from the cards ZSPm2 and ZRPf3 which show that P is an ancestor of Z's sire and also of Z's dam. Thus $2 + 3 = 5$ is entered in Table II in the cell where the row for Z and the column for Z coincide. Ancestors with only male or only female cards for a subject are ignored in figuring inbreeding; these contribute to the inbreeding of an intermediate ancestor but not directly to the inbreeding of the subject. An example is the pair of cards ZOIf3 and ZNIf4 in the third column of Table I. Although these show that I is an ancestor of Z thru two different offspring of I, the f's show that both of these lines came to Z through Z's dam. A glance at the pedigree of Z in Figure 8 confirms this and shows that this pair of lines contributed to the inbreeding of Y but not to the inbreeding of Z.

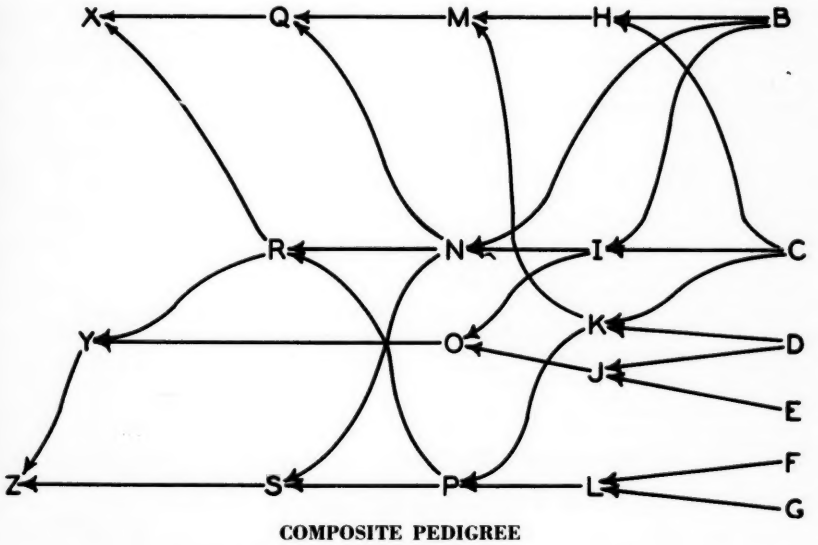
The procedure can be made entirely routine by moving down the tabulated data sheet, adding the $n + n'$ for each ancestor group, and entering them in the appropriate cells from left to right in such a two-way table as Table II. The first row of $n + n'$ in each cell of Table II comes from the first column of Table I, the second row from the second column, etc., for illustrative and checking purposes.

Inbreeding of Ancestors

Wright's formulas can be resolved into a form which avoids direct computation of F_A, the inbreeding of a common ancestor A. The formula $\Sigma [(\frac{1}{2})^{n+n'} (1 + F_A)]$ may be written $\Sigma [(\frac{1}{2})^{n+n'}] + 2 \sum_{A'} [(\frac{1}{2})^{n+n'+m+m'}]$. Here the $\sum_{A'}$ includes all paths from the subject through A to A's common ancestors, while the $n + m$ and $n' + n'$ are the corresponding numbers of generations. With the card system, only the $(\frac{1}{2})^{n+n'}$ is credited to A; the remaining $(\frac{1}{2})^{n+n'} F_A$ is included automatically when the $n + n'$ is counted for the common ancestor of A. An example in Figure 8 is that X has some inbreeding from ancestor N, but N is itself inbred ¼ to B. The inbreeding of X to N is thus $(\frac{1}{2})^3 (1+\frac{1}{4})$ in terms of Wright's original formula. The same total comes automatically from Table I in two parts: $n + n' = 4$ for the pair XRNm2 and XQNf2, while $n + m + n' + m' = 7 = n + n'$ for each of the two pairs, XNBm3 with XIBf4 and XNBf3 with XIBm4. The principle applies to computing both inbreeding and relationship coefficients.

One adjustment may have to be made in the coefficients computed by the card system. The kind of cases in which this occurs is illustrated by the arrow diagrams in Figure 9. The card system gives $n + n'$ values of 3, 8, and 8 for ZX, and 2, 7, and 7 for YX for the paths shown in Figure 9*a* but the coefficients are obviously 3 and 2 only. This situation is always traceable to *an inbred ancestor of a common ancestor*. In this case R, a common ancestor in XY and in XZ, has an inbred ancestor N. N also figures here as an inbred ancestor of Y who is an ancestor of Z, causing two spurious paths of 8, 8 to be counted for ZY. The situation occurs also in computing the $n + n'$ for ZY as shown in Figure 9*b* where the spurious paths automatically counted to C because R is an inbred ancestor of the common ancestor, Y, are 9, 9. Deletions of 9, 9, 8, 8 for ZY, 8, 8 for ZX, and 7, 7 for YX from Table II are necessary to remove these spurious paths. The ZZ, YY and XX cells need no adjustment in this particular example because no common ancestor of the sire and dam of a subject happens to have an inbred ancestor.

These adjustments can be made routinely where the inbred ancestors of common ances-



COMPOSITE PEDIGREE

Figure 10

Complete arrow diagram showing the hereditary pairs which are responsible for the inbreeding of individuals *X*, *Y* and *Z*.

tors are known. It seems easiest and most accurate first to count all of the paths listed by following a routine procedure and later to identify and delete the spurious paths. The appropriate subtraction is $\sum_{A'} (\frac{1}{2})^{n+n'} F_{A'} = 2 \sum_{A'} (\frac{1}{2})^{n+m+n'+m'}$ for both inbreeding and relationship coefficients. Here the $\sum_{A'}$ includes only paths from the subjects through A (the common ancestor) and A′ (the inbred ancestor of the common ancestor) to the common ancestor of A′ such as the B and C shown in Figure 9. Inbred ancestors of common ancestors, if not known, can be identified by constructing an arrow diagram of the hereditary paths from the printed list.

An arrow diagram of the pedigrees in Figure 8 is given in Figure 10. Since the only cases where this correction is needed are those where a common ancestor itself has an inbred ancestor, the work can be systematized by listing, as Table II is being filled, those ancestors through which each value of $n + n'$ comes. Then one need look only at the pedigrees of those listed ancestors to see if any ancestors of theirs are inbred. Their own inbreeding, if any, needs no attention, as its effects are included automatically as already described.

Final Computations

The formulas needed for obtaining numerical values for Wright's inbreeding coefficients and the genic covariances from the values of $n + n'$ in Table II are $F_X = 2\Sigma(\frac{1}{2})^{n+n'}$ and $R_{XY} = \Sigma(\frac{1}{2})^{n+n'}$, respectively. Because $n + n'$ are powers of ½, they can be reduced by simple steps such as $8 + 8 = 7$, $7 + 7 + 6 = 5$, etc. This often saves much labor in the subsequent addition. Both the reduced powers of ½ and the inbreeding coefficients and genic covariances for Z, Y and X are given in Table III. The values 8, 8 for ZX, 7, 7 for YX, and 9, 9, 8, 8, for ZY were deleted from Table II before making the reductions shown in Table III, these being the adjustments necessary for the inbred ancestors (N and R) of the common

TABLE III.—Reduced values of n + n′, inbreeding coefficients and genetic covariances.

Value	Subject	Z	Y	X
n+n′	Z	4, 5, 7, 8	1, 2, 6, 7	1, 6, 7, 8, 9
F or R		.2110	.7734	.5294
n+n′	Y		4	1, 6, 8
F or R			.1250	.5196
n+n′	X			3, 4, 5, 6, 7
F or R				.4844

ancestors (R and Y) as shown in Figure 9. Thus the genic covariance for Z and Y can be reduced to $(\frac{1}{2})^1 + (\frac{1}{2})^2 + (\frac{1}{2})^6 + (\frac{1}{2})^7 = 0.7734$. The inbreeding of Z reduces to $2[(\frac{1}{2})^4 + (\frac{1}{2})^5 + (\frac{1}{2})^7 + (\frac{1}{2})^8] = .2110$.

When an inbred line is small and is kept closed to outside blood for many generations, labor may sometimes be saved by computing by this card method the inbreeding coefficients and genic covariances between all the individuals living at a date about five to eight generations after the line was closed to outside blood and then computing these statistics for future individuals by formulas from tables corresponding to Table III. Emik and Terrill[2] have described this recently.

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TASTE-BLINDNESS AND DIABETES IN THE COLORED POPULATION OF JAMAICA

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PREVIOUS communications to the Journal of Heredity[1, 3, 4, 5] have reported an association of taste-blindness with diabetes mellitus in a population composed of 512 white persons and 168 negroes in California. 41 percent of the white diabetics were found to be unable to detect the bitter taste of phenylthiocarbamide (PTC) when presented in the usual form of filter paper impregnated with that compound.

Among 170 negroes only 13 diabetics were found: none of them taste-blind. The author therefore remarked that perhaps negroes might constitute a special case with respect to PTC sensitivity and diabetic susceptibility. An opportunity to investigate that point in the large colored population of Jamaica in the British West Indies was afforded by the John Simon Guggenheim Foundation. The present paper reports the results of that investigation.

It is proper to observe here that "colored," the term used in this paper, is the term preferred in the island. It comprises all persons whose ancestry is, wholly or in part, African Negro—perhaps 98 percent of the present population of 1,300,000.

The chi-square test renders it extremely improbable that the differences observed between the diabetics and the non-diabetics with respect to ability to taste PTC could be due to chance. ($X^2 = 12.93$; P is approximately 0.001.)

The 121 persons recorded as diabetic came from several institutions.* The 632 non-diabetics include 51 persons vaccinated at Red Hills, 92 Maroons at Acompong, servants at the hotel, etc. perhaps 250 at most. The remainder come from sources previously men-

TABLE I.—Distribution and P.T.C. taste blindness in a Jamaican population.

	+	(—)	Total	Percent Taste-blind
Diabetic	99	22	121	18.18
Non-diabetic	*583*	*49*	*632*	*7.75*
Total	682	71	753	9.44

*The majority came from the wards and out-patient departments of Kingston Public Hospital and the other hospitals which are under the direction of Dr. Fitzmaurice, Senior Medical Officer of the Island. Others were tested at like departments of the Seventh Day Adventist Hospital at Kingston, under Dr. Druitt, Mr. Kerr-Jarett of Montego Bay, Custos, made it possible for me to visit the somewhat isolated settlement of Maroons in the parish in which Acompong is situated. Mr. Kerr-Jarett accompanied me to Acompong. Mrs. March, Chief Visiting Nurse of Kingston and St. Andrew's parishes, invited me to go with her and others from the Kingston Health Center to a village on a stream about seven miles above Kingston, where 51 persons were to be vaccinated against typhoid fever (known there as "the fever".) My thanks are extended to all these people, without whose help the data could not have been collected.

tioned and are of equal validity with 121 diabetics.

The PTC paper used was supplied by Dr. Beadle, of the California Institute, the same as used previously by Dr. Segall and the writer. Dr. Harris *et al*[1], using a different method of testing, have recently reported finding 41 percent of persons with nodular goiter to be taste-blind, but only 31 percent diabetic. No other work bearing on the subject of taste-blindness in diabetes has come to the writer's attention. Findings in small groups, standing alone, can be quite misleading, and one hopes that additions will be made by others to the lists of persons with diabetes and goiter.

The method employed by Dr. Segall and the writer has been adequate, in the hands of others, to establish the Mendelian character of taste-blindness and to indicate the frequency in large groups of presumably normal people such as a college freshman class. It would seem to be, at present, the method of choice for any large-scale survey.

Nothing is known so far as the writer is aware, of the realtive frequency of diabetes in white and colored peoples. It would seem that, whatever the relation of taste-blindness to diabetes may be, the relation is of the same nature in both white and colored.

The relative infrequency of taste-blindness among colored people has been recorded by other observers, but the writer's extraordinary findings among the Maroons at Acompong deserve special attention. Here only 3 such cases were found among the 92 adults tested, or 3.26 percent. These people ascribe their origin to long-ago runaway slaves who took refuge in the "Look Behind" hills of this region, whence they sallied now and then to make a good deal of trouble. England made a treaty with them in 1735. My informant in Kingston spoke of them as follows: "They have been overglamorized; they are assimilated [with their environment]. The treaty is no longer in effect. It is true, however, that they retain certain privileges and customs and preserve a feeling of separateness." In short, one may say they are somewhat nearer the original stock.

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MARCUS CLAUDE TERRY—1871-1950

DR. TERRY died suddenly of acute coronary occlusion on September 6, 1950 at the age of 79 at Salem, Oregon, while on a motor trip in the Northwest. Thus ended the life of a man of wide interests and remarkable enthusiasm for scientific questions which caught his fancy. This is reflected by the present paper—his last—dealing with the phenomenon of taste blindness, work which was made possible by the award to him recently of a John Simon Greggenheim Memorial Fellowship. It is highly significant of the worth of the man, and of the merit of this his last research project that he was awarded this fellowship at such an advanced age. No young scholar embarked on a scientific mission with more enthusiasm than did Dr. Terry on the trip to Jamaica to make the observations here reported.

Dr. Terry was graduated from Rush Medical College in 1899. Following his graduation he served on a part-time basis as clinical instructor in Immunology at Stanford University. He served in the Army during World War I and following this on the staffs of various Veterans Administration Hospitals. He was primarily interested in clinical pathology and his positions were generally associated with the laboratory in the hospitals which he served. Though much of his life was spent on routine work, he was no mere "routinist." He is the author or co-author of 25 or more publications, mostly in the field of clinical pathology. However, to me his deep interest in things scientific is reflected best by his eagerness to come into the laboratory following his retirement from the Veterans Administration, and his happiness on being given bench space in our department. For several years he was more or less regularly at his bench working on such problems in bacteriology as could be persued as time and health permitted. We shall long remember him for his friendliness, enthusiasm, and in general his sterling qualities as a man.

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Evolution and Variation in Plants

The need for a book integrating the accumulated reservoir of facts from the various plant sciences, with current evolutionary thought, has long been overdue. Prof. Stebbins is exceptionally well qualified to do the required job of integration and interpretation.

For over fifteen years he has been an active fact-gatherer in two of the largest and most scientifically interesting families of higher plants, namely, the Gramineae and Compositeae. Besides, he has done important cytogenetic and taxonomic research with such genera as *Paeonia, Solanum, Sequoia,* and others.

For the above reasons, it is doubtful whether a better informed person than Stebbins could have been found to state the case for evolution and variation, provided by the evidence from the plant kingdom. He has assembled and digested an enormous amount of material.* Furthermore, he has assessed its value and interpreted its evolutionary significance. Although Stebbins is not bashful about risking his personal opinions on controversial subjects, he is equally careful to record the evidence from all sources before doing so.

The chapter headings given below are indicative of the wide range of subject matter the author has been able to cover in this book. The first four chapters are concerned with the several aspects of variation. These are followed by chapters on "Genetic systems as factors in evolution," "Isolation and the origin of species," and "Hybridization and its effects." Then follow two chapters on polyploidy, one on apomixis, one on structural hybridity and the genetic system, and two chapters on evolutionary trends, followed by a final chapter labelled, "Fossils, modern distribution patterns, and rates of evolution."

It is obviously impossible to review in detail the many fine features of the book, but at least three topics deserve additional comment. Stebbins has recognized the value of the carefully designed experiments of Harlan and Martini with barley. This work has furnished some of the best experimental evidence with respect to the operation of competition and natural selection in cultivated plants.

The younger generation of biologists will have reason to be grateful to Stebbins for pointing out, and evaluating the importance of the early work done in Russia (Zinger, 1909; Sinskaia and Beztuzheva, 1921), and Sweden (Tedin, 1925) on the behaviour of species of the cruciferous genus *Camelina,* in cultivated flax fields. This study presents a convincing analysis of the factors that are effective in bringing about the truly remarkable adaptive effects that can be achieved under the guidance of natural selection.

The author has had the courage to tackle the difficult problems involved in the differentiation of the higher categories of the plant kingdom (orders, families, genera). Because of the nature of the evidence, this is perhaps the least satisfactory portion of the book, and many systematists will be unhappy with the conclusions that emerge. Nevertheless, Stebbins must be credited with suggesting areas where certain of these problems are subject to an experimental approach, and he has devised an ingenious survey technique that leads to some interesting correlations when applied to families of the angiosperms.

Stebbins believes that, "all the trends leading to the differentiation of families of flowering plants probably took place simultaneously and at a relatively early stage in angiosperm evolution." The plant distribution picture and the evidence from paleontology suggest that separation into families was complete at a comparatively early date, probably not later than the Cretaceous Period.† With these twin ideas as a starting point the author has proceeded to erect a tentative hypothesis for the differentiation of the families and orders of the flowering plants. It is assumed that the earliest appearance on the species level of characteristics now considered to be diagnostic of families and orders was largely the result of chance, but the fabrication of successful combinations of characters to form these higher categories was guided mostly by natural selection. In the words of the author this hypothesis, "suggests that natural selection has played a large role in directing evolutionary specialization during the differentiation of the families of flowering plants, but that this role has been obscured by the effects of chance in the first establishment of individual characters, by parallel evolution, and by hybridization."

For a book of 643 pages there are surprisingly few typographical errors. Several mechanical slips have occurred, but these are not serious. Some of the figures would have been more meaningful, had the reproduction been neatly done. The "Works Cited" list contains over 1200 citations, and there is a useful and accurate index.

Clearly, this is a book that could be read with profit by all biologists. It is not just another compilation of the literature, but a carefully planned, and scholarly synthesis of the significant phenomena responsible for evolution and variation in plants.

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**Evolution and Variation in Plants.* By G. Ledyard Stebbins, Jr. i-xix, 1-643, f. 1-55; t. 1-9. Columbia University Press, New York. 1950.

†This assumption is supported in a recent paper by Sussenguth (Sussenguth, K. "The flora of Australia as a measure of the antiquity of the Angiosperms." *Pacific Science* 4:287-308, 1950.) He concludes from indirect evidence supplied by an analysis of endemism in the Australian flora that the development of the primitive families of angiosperms must have taken place during the Middle and Lower Cretaceous, or possibly even earlier during the Jurassic period. However, it is admitted that this conclusion has not been confirmed by fossil evidence.